

# The Road Towards an HIV Cure

**Authors: Craig McClure and Darien Taylor**

October 2017

**We have done our best to write in plain language. However, an HIV cure treatment glossary may help you to read this article. We recommend *Project Inform*'s: <https://www.projectinform.org/hivcureglossary/>**

## I. Introduction

In recent years, a global movement has arisen towards finding a cure for HIV. While we remain many years away from a cure that could be offered to the almost 37 million people living with HIV throughout the world, researchers are exploring a number of approaches that might one day bring an end to the need for people living with HIV to remain on lifelong antiretroviral therapy (ART).

On the two days preceding the International AIDS Society (IAS)'s 9th HIV Science Conference, which took place in Paris in July 2017, a meeting was held involving *Towards an HIV Cure*, an international scientific initiative formed by the IAS in 2011, and leading cancer researchers from around the world. The meeting focused on sharing recent advances in the fields of HIV and cancer that could prove beneficial to the search for a cure for both diseases.

The purposes of this report are:

1. to define what is meant by “cure” in the context of HIV and to outline the basic background concepts of HIV cure research;
2. to share the most promising research findings presented at the pre-meeting and the main IAS conference related to the search for an HIV cure;
3. to outline the major issues specific to the HIV community that must be resolved to accelerate research towards finding an HIV cure, and;
4. to present options for community members to learn more and/or get involved in the movement towards an HIV cure.

This report is aimed primarily at a community audience, including people living with HIV, their sexual partners and allies.

## **II. Background Concepts**

### **a) What is an HIV “cure”?**

Any strategy or combination of strategies that would eliminate HIV from a person’s body, or permanently control the virus and render it incapable of causing disease, can be referred to as a “cure”. A “sterilizing” cure would completely eliminate HIV from the body, whereas a “functional” cure would keep HIV viral load permanently suppressed, or undetectable, without the use of ART, thereby preventing it from causing damage to the immune system that ultimately results in the illnesses collectively known as Acquired Immune Deficiency Syndrome (AIDS). The global HIV community of researchers and advocates has borrowed the word “remission” from cancer research to describe this state of highly potent and long lasting successful treatment.

### **b) Has anyone ever been cured of HIV?**

Yes. The first and only person known to have achieved a sterilizing cure of his HIV infection is Timothy Brown, who is also known as the “Berlin patient”. Diagnosed with HIV in 1995, he was treated with ART for ten years before being diagnosed with acute myeloid leukemia (AML). Following a standard approach to treating AML, Brown received radiation and chemotherapy to wipe out his immune system, then was given a stem cell transplant to rebuild it. The donor who provided the stem cells was immune to HIV, because of genetic mutations he had to CCR5, a protein that allows HIV to enter blood cells and cause HIV infection. About 1% of Caucasians carry this genetic mutation. When Brown received the stem cell transplants, his re-built immune system was immune to HIV. For over ten years, he has been off ART and doctors have been unable to detect HIV in his body.

### **c) Why is this cure not given to everyone living with HIV?**

Stem cell transplants are extremely risky and only given as a last resort to people with leukemia and other advanced cancers. Brown himself required two stem cell transplants to cure his leukemia and he almost died after the second transplant. Stem cell transplants are also very expensive.

A number of other people have received similar procedures, though the exact procedure has never been repeated successfully. It is very difficult to find donors who are available for transplantation and who also have the CCR5 mutation. In a study in Boston, 2 HIV infected people who needed transplantation received stem cells transplanted from donors without any mutation in the CCR5 gene. In spite of radiation and chemotherapy and restoration of the immune system with stem cells from an uninfected person, their HIV rebounded. Thus, HIV-infected cells remained despite radiation and chemotherapy and the virus was able to infect new cells after transplantation. Although these procedures failed to achieve an HIV cure in individual participants, they were important in: a) assisting research to understand that reservoirs of HIV-infected cells persist after radiation and chemotherapy; and, b) understanding the importance of CCR5.

Last year for the first time, a European research consortium reported that three volunteers who have undergone similar cancer treatments have survived the treatments and appear to be virus free or controlled. However, these individuals are still on ART and no claims have been made that they are in viral remission. Further monitoring is required, as well as the agreement of the individuals to go off ART, to know if they are indeed free of HIV.

### **d) Why is ART so important, but not enough to cure HIV?**

Up until 1996, almost everyone living with HIV died of AIDS within a few years of infection. The discovery that combining three or more antiretroviral drugs could control HIV from infecting and destroying active cells meant that HIV infection can become a manageable chronic condition. Since then, most people who are able to take daily ART remain alive and well and can live a near-normal lifespan.

It is now known that the earlier a person begins taking ART after HIV infection, the better. The longer HIV remains uncontrolled in the body, the more damage it can cause. In the early hours, days and weeks of HIV infection, the virus infects cells that are resting, or inactive, in areas of the body that ART is unable to get into. The areas of the body where HIV can hide out inside resting cells that are not producing new copies of HIV are called “latent reservoirs”. These are predominantly in the gut, the brain, the lymph nodes, and other tissues and organs. Less than 2% of these latent reservoir cells are found circulating in blood. Yet to date in research settings, many reservoir measurements have taken place solely in blood samples that are easy to obtain. Invasive biopsy samples of organs and tissues are hard to get.

As long as a person takes ART, the current consensus is that the HIV in latent reservoirs may do little damage in the long term. However, this consensus is not absolutely demonstrated, since tiny levels of HIV replication that cannot be measured may be continuing in these areas of the body. However, as soon as most people stop taking ART, the HIV infected cells in the reservoirs become activated and produce more HIV, causing viral load to increase and disease to further progress. This is why ART alone is not enough to cure HIV.

There is a global consensus that the earlier someone starts ART after HIV infection, the harder it is for HIV to establish latent reservoirs. People are encouraged to seek HIV testing immediately after they think they may have been infected and even to seek post-exposure prophylaxis (PEP) within a few hours after an exposure/ potential exposure to HIV to try to prevent HIV infection itself. In addition, the option for HIV negative people who are at risk of infection to take antiretrovirals regularly to prevent HIV infection, called pre-exposure prophylaxis (PrEP), also now exists.

Researchers around the world have brought together groups, called cohorts, of people living with HIV who began ART very early after HIV infection. Several people in these cohorts have agreed to be in clinical trials where they stop ART after a certain period of time (a treatment interruption which is referred to as an “analytic treatment interruption” or ATI in the context of a clinical trial) to see whether HIV rebounds, sometimes after receiving other experimental therapies. So far, only a few people have not rebounded after a few weeks or months off ART. None of these individuals is considered cured and no one has been followed long enough to know if control of HIV is due to ART or their own immune system.

### **e) How can babies living with HIV help us find a cure?**

For pregnant women and nursing mothers living with HIV, ART is an effective way to stay healthy and prevent HIV transmission to their infants, either during pregnancy, during delivery or through breastfeeding. Unfortunately, not all pregnant women and nursing mothers are aware of their HIV status or have access to ART. In 2016, about 160,000 children were infected with HIV worldwide, a number that has been declining as more pregnant women across the globe access ART.

Babies living with HIV offer important clues towards developing an HIV cure. Children's immune systems are not fully formed until the age of four or five. An infant does not start to produce antibodies efficiently for the first few months of life. It may therefore be more difficult for HIV to establish latent reservoirs in babies. Providing ART to infants immediately after diagnosis of HIV infection is not only crucial for keeping the babies alive and well, but may also be easier to achieve a functional HIV cure than for adults. Cohorts of infants living with HIV are playing an important role in HIV cure research.

In 2013, a little girl known as the "Mississippi Baby" was thought to have been cured of HIV. She was treated with ART within a few hours after birth, but treatment was stopped at the age of 18 months the child was lost to follow-up in the health care system and she no longer received treatment. Five months later, when the child was next seen by doctors, they could find no detectable HIV in the girl and HIV could not be detected in her for the next two years. However, HIV rebounded after 27 months. The girl once again began ART and now has undetectable virus. She remains on ART. While many questions remain, her experience is stimulating research into the value of timed ART, delivered at the earliest possible time point after HIV infection.

### **f) More on Latent Reservoirs and the Road to a Cure**

Finding a way to eliminate latent HIV reservoirs in people living with HIV is the "holy grail" of HIV cure research. Many approaches now being tested towards finding an HIV cure involve either "shocking and killing" (also known as "kicking and killing") HIV-infected cells in latent reservoirs using latency-reversing therapies or "blocking and locking" those cells. Other approaches include ways to make cells resistant to infection, to remove their susceptibility to infection, or to use antibodies and other means to

allow immune functions to work against the virus even without kicking or locking. The remainder of this paper addresses the most recent of these research efforts, a potential way to standardize the measurement of the latent reservoir, and lessons to be learned from recent advances in cancer research.

### **III. Highlights from the 9<sup>th</sup> IAS Conference on HIV Science, July 23-26, 2017 and the IAS HIV Cure and Cancer Forum, July 22-23, 2017**

#### **a) Better understanding the HIV reservoir**

Research was presented at both IAS 2017 and the HIV Cure Forum suggesting that diminishing the size of the latent reservoir with new types of treatment at the onset of acute infection in at least some individuals may prolong the time of HIV remission in the absence of ART. However, time of HIV remission after ART is withdrawn appears to also depend on the ability of the individual's immune system to naturally control HIV. This topic will be explained further in Section c) below ("Improving Host Ability to Respond to HIV: Gene Therapy"). Eliminating the latent reservoir is a daunting task, but if research was able to discover how this could be done, HIV could be cured. It is important to note, however, that the notion that reservoir reduction is a necessary component of remission remains a plausible but unproven hypothesis.

Recent research strategies have involved using new types of latency-reversing drugs to "shock", "kick" or "flush" out and then eradicate HIV-infected cells which are latent in HIV reservoirs. (See more information on the "kick and kill" approach in Section e), below.) However, these latent cells are currently very difficult to identify, due to their location in the reservoir. Without being able to differentiate latent HIV-infected reservoir cells from uninfected cells, there is no way to specifically deliver drugs to target infected cells. Being able to better target the delivery of these latency-reversing drugs would reduce their toxicity and side effects, which currently present huge disincentives to enrolling people with HIV in clinical trials for cure-related therapies.

One of the most significant pieces of research presented at both conferences concerned the recent discovery of a marker by which HIV reservoir cells can be identified. Two different studies discussed recent findings that HIV-infected reservoir cells express much higher levels of a cellular receptor molecule called CD32a than surrounding cells. In addition, research characterizing the viral reservoir shows that CD32a is found on more mature immune cells, and CD32a+CD4 cells have greater than 100-times more HIV DNA than CD4 cells without CD32a. CD32a+CD4 cells have high levels of immune checkpoint markers on their surface. These results confirm the importance of CD32a as a marker of the HIV reservoir. If a simple test could be developed to measure CD32a in the blood, individuals participating in clinical trials of experimental cure approaches might no longer require invasive biopsies of tissue samples to determine the presence of HIV in latent reservoirs.

It is important that HIV cure research continues to better characterize the HIV reservoirs in order to develop and assess the effect of innovative experimental strategies, such as the two-step “kick and kill” approach, when they are given to people with HIV in clinical trials. The development of new technologies to estimate the size of the viral reservoir will help to achieve a more accurate estimate of the amount of HIV that is capable of restarting systemic HIV infection after ART is withdrawn, and the estimated time to systemic HIV infection or viremia. Cure research also needs to clarify how the HIV reservoir is initially established after HIV infection, in order to understand how to prevent the initial establishment of reservoirs after people are first infected. Research also needs to tell us more about what happens when people with HIV who have an undetectable viral load but well-established HIV reservoirs experience treatment interruptions in clinical trials of innovative new therapies. What might characterize their experience of rebounded viral load after sustained viral remission, and how might therapies be developed to stop the redevelopment of viral reservoirs in people treated by a “shock and kill” approach?

Such research questions also point the way to new strategies for the prevention of HIV acquisition by developing therapies to block the initial “seeding” of reservoirs with HIV. These questions also have implications for the development of innovative HIV treatments, by developing therapies which specifically flush HIV-infected cells from the reservoirs and prevent the redevelopment of reservoirs in the case of viral rebound. And finally, these questions about the reservoir may lead in the direction of a cure for HIV through the development of effective combination cure therapies which deliver on the promise to “kick and kill” HIV in the body.

## **b) Early treatment intervention in acute HIV infection**

The short period of acute HIV infection immediately after HIV has entered the body appears to provide a window of time in which - with the development of better therapeutic interventions - an individual's immune system might be “tweaked” to more effectively combat HIV. Understanding more about what is going on in the immune system in early HIV infection and how to create “better” immune responses against HIV in people who are recently infected is an important step in improving treatment for HIV, moving toward “sustained viral remission” without therapy and creating a vaccine against HIV.

For a number of years, the French VISCONTI trial has followed a small cohort of people with HIV who started treatment shortly after becoming infected and who later stopped treatment for various reasons. The participants being followed in this cohort have subsequently exhibited viral control on standard tests for an average of seven or more years. This cohort study seems to show that early treatment may lead to sustained viral suppression in at least some people, although the underlying explanation for these observations remains unclear.

IAS 2017 delivered the news of an HIV-infected child in South Africa who is controlling the virus without ART. The child was born to an HIV-infected mother and was given ART starting at 8 weeks of age. The treatment was stopped at 40 weeks as part of a prospective controlled clinical trial. Now, more than eight years later, the virus has not rebounded. However, the child still has low levels of HIV that are easily detected with ultrasensitive tests.

To date, little is known about the mechanisms of sustained HIV remission. It may be a delicate balance between using drugs early enough to keep the HIV reservoir small, but not so early that the immune system is not able to encounter enough HIV to develop a good memory response to be used if and when the virus rebounds. The role of individual genetics to boost this control is still being studied. If effective treatment of HIV is started very early (i.e. within hours after infection), it is possible that only a few hundred cells may be infected, but the immune system may not have had a chance to generate a protective response. On the other hand, if treatment is started too late, recovery of immune function may be too difficult. The South African child, the Mississippi Baby, the French VISCONTI patients, and others may hold the evidence that will help research to better characterize the right balance and which may assist in the development of new therapies.



Upcoming Thai clinical trials will hopefully answer the question of when to treat during acute HIV infection for the purposes of creating an effective immune response that will permit long periods of sustained viral remission. These clinical studies which are designed to reduce viral reservoirs and increase immune control during hyper-acute HIV-infection. Another study during hyper-acute HIV infection, the FRESH study, is also underway in South Africa. The results of these studies will strongly inform understanding about whether manipulation of viral reservoirs and immune responses during the earliest stages of viral infection does indeed translate into beneficial effects such as sustained HIV remission after the withdrawal of ART.

### **c) Improving Host Ability to Respond to HIV: Gene Therapy**

Gene therapy aims to identify genetic characteristics that help to naturally control the virus in some people living with HIV known as “elite controllers” or “long term non-progressors” and to develop mechanisms for transferring these genetic characteristics to others who do not have that ability. This strategy may have its limitations since elite controllers may still experience inflammation that enhances other disease risks.

One of the main targets for research in the field of gene therapy is the CCR5 receptor – a crucial entry point for HIV to infect healthy CD4 cells. The donor who provided stem cells for the transplant that ultimately cured Timothy Brown had genetic deletions to CCR5, making it impossible for HIV to enter and infect CD4 cells. Scientists are exploring ways to manipulate genes in order to turn off the CCR5 receptor.

This strategy is being researched in three different ways, all of which require the genetic alteration of immune cells. The first strategy, preventative in nature, aims to genetically alter cells so that HIV cannot infect them. The genes of the immune cell are edited to remove CCR5, the mechanism used by HIV to enter and infect CD4 cells. A second strategy would enable immune cells to find and fight HIV more efficiently. The immune cells are genetically modified to better detect HIV-infected cells in latent reservoirs in the body. The third approach is the most complex, and involves removing or “editing” HIV from the DNA of infected cells.

Gene therapy involves extracting immune cells from HIV-positive individuals and then genetically modifying them. There are many challenges in this area of cure research. It has been difficult to detect and extract resting white blood cells infected with HIV, i.e. one of the components of the latent HIV reservoir. The identification of the CD32a marker is important, as it will assist the development of gene therapy that can identify these latently HIV-infected immune cells. The quantity of cells with modified genes needed to significantly improve a person's ability to control HIV without drug therapy is as yet unknown. Researchers are working on developing a method to deliver gene editing technology directly into the body. The hope is that this approach will quickly spread modified genes through the whole body.

Editing host genes to better respond to or block HIV would be an incredibly powerful tool. The Cure Forum heard more news about promising gene-editing strategies used for treating cancer, including chimeric antigen receptor (CAR) T cells, which are now being adapted as a means to potentially control HIV. Gene editing strategies could fundamentally change our susceptibility to HIV.

#### **d) Learning from Cancer: Targeting Immune Checkpoint Receptors**

Immune checkpoints are molecules on the surface of cells that either “turn up” or “turn down” (accelerate or inhibit) the strength of a molecular signal sent by the immune system to disease-controlling cells. HIV infection, like many cancers, is able to disrupt the immune system's efforts to prevent or minimize the effects of infection, by turning down the T cell signal that tells the immune system to do its work.

HIV-infected cells are particularly rich in these immune checkpoint receptors, as are cancer cells. Current research aims to develop drug therapies that target immune checkpoint receptors which prompt the cell to stop whatever immune job it is doing and revert to its latent state. A few drugs to target these molecules are already available to treat some forms of cancer.

There are a number of different immune checkpoint molecules, with names such as CTLA-4, PD-1, TIGIT and LAG3. Drugs which inhibit these immune checkpoints receptors are being developed for use in HIV remission studies. They are called “checkpoint inhibitors” or “blockers.” The HIV Cure and Cancer Forum heard about several experiments using these agents in animal models or in cancer patients

with HIV, with drugs such as pembrolizumab, nivolumab, baracitinib, rinoxolitinib and others. These drugs may have potential to be used in the future as an addition to ART to slowly shrink the HIV reservoir to the point where a treatment interruption could be considered.

To date, most results of using CTLA-4 and PD-1 inhibitors in people with cancer who also have HIV have not been impressive, with only a few individuals able to interrupt their HIV treatment for a short period of time. It is difficult to draw conclusions from cancer-focused research about how these drugs may work more generally in people with HIV who do not have cancer, and currently these drugs are so toxic that they are unlikely to attract clinical trial participation by people with HIV who are doing well on ART. However, the immune checkpoint inhibitor PD-1 was the subject of a plenary session at the IAS Conference. The speaker, Dr. Tasuku Honjo from Japan, was very positive about the role that PD-1 could play in HIV cure research. PD-1 inhibitors have been a recent game-changer in the treatment of a number of advanced stage cancers. Dr. Honjo stressed the need for clinical trials of PD-1 inhibitors in people with HIV and predicted that they may be able to create a functional cure for HIV by inducing sustained viral remission. If short term PD-1 therapy proves successful for HIV, it may also prove to be more cost-effective and have fewer side effects than lifelong ART. At present however, PD-1 blockade strategies remain risky and unproven.

### **e) From “shock and kill” to “block and lock”**

The “shock and kill” HIV cure strategy, which proposes the use of “latency reversing” drugs, has received attention in recent years. This cure strategy aims to flush (or shock, or kick) the virus out of latent reservoir cells into activity in the bloodstream using latency-reversing agents. Once the infected cells are activated, other drugs—perhaps a yet-to-be discovered therapeutic vaccine or antibody-based therapies which would harness the body’s own response to HIV—would then kill the HIV-infected active cells. The harnessed response might be sufficient even without flushing out the virus.

Some of the latency-reversing agents being considered are currently used as cancer treatments, although researchers are also trying to discover new drugs. One of the agents currently under investigation is called a Histone deacetylase (HDAC) inhibitor. However, repeated experiments with different HDACs have shown that - while they certainly wake up reservoir cells and turn them into short-lived virus-

productive ones - they are unable to prevent new cells being ‘seeded’ with HIV and then returning to latency in a reservoir. The size of the latent reservoir cells therefore does not change significantly.

The Cure and Cancer Forum suggested that keeping the viral reservoir in a state of permanent lockdown may be an alternative to the “kick and kill” strategy. This would involve the discovery of a drug to detect the few HIV-infected cells that exist in the body of a person who is virally suppressed on ART, and then, rather than shock and kill the cells, do something to the DNA of these latent cells so that they are never reactivated. The DNA of these cells could be “methylated” or permanently immobilized. In order to not damage the overall DNA structure of these cells, only the parts which surround the HIV genes would need to be immobilized. It is currently very difficult to know where exactly HIV has integrated itself along the genome of the cell, but research is beginning to describe the reservoir more accurately and uncover markers like CD32a, by which infected reservoir cells can be identified and targeted for therapy.

At IAS 2017, data on this “block and lock” strategy were presented from experiments in mice of an oral inhibitor of the HIV protein called tat. Antiretroviral drugs in combination (ART) block different steps of the HIV replication cycle (reverse transcription, integration, protease and others). ART does not prevent HIV transcription. That is a reason why Tat inhibitors may be important. They inhibit one step in the HIV replication cycle that is not suppressed by current ART.

Tat is one of the first proteins expressed by the virus after infection and sets in motion other events, including HIV transcription. When HIV genes are integrated in the genome of the host cells, Tat protein activates the transcription of the HIV gene, leading to the formation of HIV. Tat inhibitors silence the virus by inhibiting the transcription of the virus, in turn preventing the formation of new HIV and the infection of cells. Tat helps to keep latent cells latent. It also appears to play a role in maintaining the slow, ‘behind the scenes’ ongoing replication of HIV that keeps the reservoir topped up.

Earlier lab research conducted on human cells has shown that the tat inhibitor, didehydro-cortistatin A (dCA), greatly reduced viral expression in HIV-infected reservoir cells. This effect seemed to last for weeks to months, even when dCA therapy was stopped, indicating that the drug had produced a persistent state of lockdown in the cells. Human studies are planned.

## IV. Community Discussion

It is not too early for the HIV community—people with HIV, their partners and allies, ASO workers, clinicians, policy makers and, of course, HIV researchers across disciplines -- to begin linking their current practices in HIV to the possibility of an eventual cure for HIV. This does not mean that a cure is around the corner, but that a body of information is beginning to emerge that has implications for what we are doing (or not doing) today in clinical care of HIV.

The relationship between the period of acute infection and early treatment intervention is important in terms of providing people who test positive now with the best possible care. Efforts to preserve immune function and retard immune deterioration now are essential if remission concepts are to be successfully implemented in people soon after HIV infection. The opportunities, knowledge, networks and technologies for very early diagnosis of HIV must be in place so that people tested in the acute phase of HIV infection can be offered therapies to reduce or prevent the seeding of latent reservoirs with HIV-infected cells. The now widely known benefits of early diagnosis and treatment for all people living with HIV may begin to be discussed in terms of research towards a cure for HIV.

Although it is important to manage expectations about the potential for an HIV cure, research has reached a point where people with HIV must be involved in the broader discussion. Plain language information resources for people who wish to know more about cure research and for those considering participation in clinical trials is essential. Standardized research protocols need to be developed with community input, both to accelerate new research and to ensure that it is ethically conducted. The HIV community must also consider how it will manage media reporting on HIV cure research, which tends to portray incremental changes as “scientific breakthroughs”.

People with HIV must seriously consider whether they are willing to be involved in and support HIV cure research. At the Cure and Cancer Forum, patient advocate Michael Louella described the need for community and researchers to identify and recruit “pozmonauts”, HIV positive people willing to take a pause from their HIV drugs in the name of science. This will likely mean that people who are doing well on their current antiretroviral combination, have undetectable viral loads and are experiencing the associated benefits, will need to consider whether they would like to get involved altruistically in cure research. This would involve using new experimental drugs or procedures through a clinical trial, dealing with the possible toxicities and side effects of these experimental procedures, and then going off

these and ART to see how long it takes for the virus to rebound and how the body reacts to these conditions. Trials with researcher-supervised analytic treatment interruptions are likely to characterize future cure research. And, since analytic treatment interruptions (stopping ARTs) are likely to be a central element of research towards a cure for HIV, it is important to develop ethical guidelines for these interruptions. There are many unknown risks about the effects of different treatment interruption designs.

The global movement towards finding a cure for HIV is growing. While a cure is years away, a body of research is emerging that has implications for what we are doing (or not doing) in our HIV-impacted lives today. This research will have implications for what we know about HIV prevention, HIV testing and diagnosis, the treatment of HIV with new therapies and finally, a possible cure for HIV.

People living with HIV and our allies need to learn more about this what is happening along the road to the cure of HIV in order to determine how best we can contribute. Our perspectives are unique and important, and need to be heard in the development of strategies that might one day bring an end to the need for people living with HIV to remain on lifelong antiretroviral therapy (ART).

*With many thanks to Ian Grubb, John McCullagh, Delphine Planas and Robert Reinhard who reviewed this work for us.*

**For more information:**

The Canadian HIV Cure Enterprise (CanCURE) is a research collaboratory focused on studying HIV persistence and developing strategies towards a functional HIV Cure. [cancurehiv.org](http://cancurehiv.org)

AVAC: Global Advocacy for HIV Prevention has exceptional community-friendly resources on HIV cure research at <http://www.avac.org/prevention-option/cure>

There are a number of articles related to HIV cure research on the website of the Canadian AIDS Treatment Information Exchange (CATIE): <http://www.catie.ca/en/treatment/hiv#curesearch>

For resources from IAS 2017 in Paris, go to [ias2017.org](http://ias2017.org)

Talk to your doctor or ASO service provider.